

## Chapter 1: Introduction

### 1.1 Background

The Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA, 1980) (CERCLA or Superfund) was enacted to provide a program for identifying and responding to releases of hazardous substances into the environment. The Superfund Amendments and Reauthorization Act (SARA, 1986) was enacted to strengthen CERCLA by requiring that site clean-ups be permanent, and that they use treatments that significantly reduce the volume, toxicity, or mobility of hazardous pollutants. The National Oil and Hazardous Substances Pollution Contingency Plan (NCP) (USEPA, 1985; USEPA, 1990) implements the CERCLA statute, presenting a process for (1) identifying and prioritizing sites requiring remediation and (2) assessing the extent of remedial action required at each site. The process includes performing two studies: a Remedial Investigation (RI) to evaluate the nature, extent, and expected consequences of site contamination, and a Feasibility Study (FS) to select an appropriate remedial alternative adequate to reduce such risks to acceptable levels. An integral part of the RI is the evaluation of human health risks posed by hazardous substance releases. This risk evaluation serves a number of purposes within the overall context of the RI/FS process, the most essential of which is to provide an understanding of “baseline” risks posed by a given site. Baseline risks are those risks that would exist if no remediation or institutional controls are applied at a site. Thus, the baseline risk assessment (BRA) provides the foundation from which DOE risk assessors can determine the most appropriate options for remediation.

Over recent years the U.S. Environmental Protection Agency (EPA) has developed a considerable body of guidance about developing baseline risk assessments for uncontrolled hazardous waste sites. But the manner in which EPA’s BRA guidance is interpreted and applied can have a great impact on the risk estimates obtained and, in turn, on the remedial option selected to reduce those risks. In general, lower baseline risk estimates should necessitate less costly remedial options than will higher risks. This document was written to (1) guide risk assessors through the process of interpreting EPA BRA policy and (2) help risk assessors to discuss EPA policy with regulators, decision makers, and Stakeholders as it relates to conditions at a particular DOE site.

This guidance provides detailed insight into the science policy issues underlying the CERCLA baseline risk assessment process as it is implemented today by EPA. It also provides the current EPA position on each issue and, through the historical perspective, explains how/why this position has changed over time. This guidance outlines the pros, cons, weaknesses uncertainties, and policy areas where more than one interpretation may be acceptable for each science policy issue. It identifies reference materials for each issue to provide risk analysts with further understanding of these issues, enabling them to both analyze their relevance and to intelligently discuss them with regulators.

The following discussion presents an overview of the CERCLA baseline risk assessment process. Introduction of the analytical components of a risk assessment will provide the framework for subsequent discussion of relevant issues. The risk assessment section will be followed by a discussion of the statutory and regulatory requirements for and key guidance related to the baseline risk assessment process. The final section of this chapter explains the structure of the remainder of this guidance document.



## 1.2 Baseline Risk Assessment: an Overview

### 1.2.1 Introduction

Figure 1.1 broadly outlines the major elements of a site assessment and remediation process and shows how these elements relate to each other. Site information development activities are conducted to make an initial evaluation of the severity of contamination at the site and to guide subsequent activities, including data gathering for the baseline risk assessment. The four steps of a baseline risk assessment are data collection and evaluation, exposure assessment, toxicity assessment and risk characterization. The results of the baseline risk assessment provide a basis for selecting an appropriate remedial option for subsequent implementation at the site. Each component of the overall process is interdependent on the others in that, if information is lacking or of poor quality in one of the components, then the entire process will suffer and produce results with higher uncertainty.

This document assumes that data collection and evaluation have already been performed, and issues related to that topic are not included herein (for completeness in this overview, however, a brief summary of the data collection and evaluation process is provided below). Subsequent chapters of this guidance focus on the three remaining components of the BRA process: exposure assessment, toxicity assessment, and risk characterization. The information for this section was obtained from the Risk Assessment Guidance for Superfund (RAGS) (USEPA, 1989).

### 1.2.2 Data Collection and Evaluation

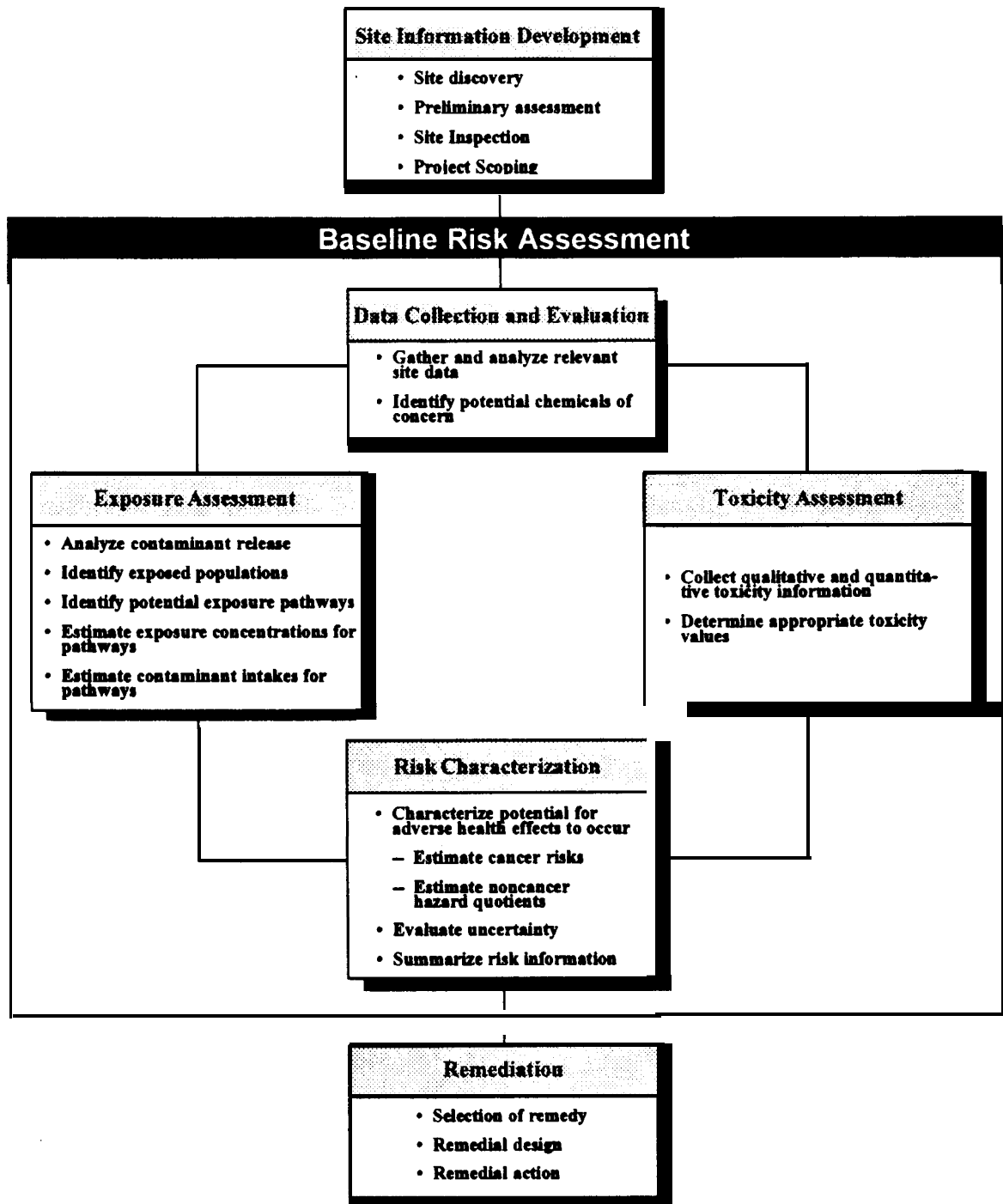
Data collection and evaluation, the first component of a baseline risk assessment consists of gathering and analyzing relevant site data and identifying potential chemicals of concern. The types of data essential for the baseline risk assessment are (USEPA, 1989) as follows

- contaminant identities,
- contaminant concentrations in the key sources and media of interest;
- characteristics of sources, especially information related to release potential; and
- characteristics of the environmental setting that may affect the fate, transport, and persistence of the contaminants.

The risk assessor must identify data needs early in the RI/FS process so that the appropriate samples can be incorporated into a sampling and analysis plan. To assess their data needs, risk assessors must have a preliminary understanding of the current and future contaminant sources, fate, and exposure routes. The risk **assessor must** also have a preliminary idea as to what predictive model(s) may be used in the risk assessment. My model parameters that require site-specific data should be addressed in the sampling and analysis plan. The sampling and analysis plan must also include the appropriate number and placement of background samples, along with blank samples.



Figure 1.1  
RI/FS Process



Source: Adapted from USEPA (1989)



Once collected, site data should be separated by medium (e.g., soil, water, air) and then evaluated. The evaluation should include analysis of the sampling and analytical method(s) employed and the quality of the data with respect to quantification limits, QA/QC measures, etc. After the above steps have been completed a tentative list of compounds upon which subsequent evaluations will focus can be established, and a corresponding data set can be chosen to be used in the risk assessment.

### 1.2.3 Exposure Assessment

Exposure assessment is the second component of a baseline risk assessment. The objective of the exposure assessment is to estimate the type and magnitude of human exposures to the chemicals of potential concern that are present at or migrating from a site. The exposure assessment process begins after the chemical concentration and location data have been collected and validated and the chemicals of potential concern have been selected. The results of the exposure assessment are combined with chemical-specific toxicity information to characterize potential risks.

Exposure, as currently in use, is defined as the contact of a chemical or biological agent with the outer boundary of an organism (USEPA, 1992). The magnitude of exposure is determined by measuring or estimating the amount of an agent available at the visible exterior of a person (i.e., the skin, mouth, nostrils) during a specified time period. Exposure assessment is the determination or estimation (qualitative or quantitative) of the route, frequency, duration, and magnitude of exposure. Exposure assessments may consider past, present, and future exposures, using varying assessment techniques. Generally, Superfund exposure assessments are concerned with current and future exposures.

The exposure assessment process consists primarily of three steps: characterizing exposure setting, identifying exposure pathways, and quantifying exposure. These three steps are described below.

Step 1: Characterization of exposure setting. Important elements of the exposure setting include the physical features of the site and pertinent aspects of the populations on and near the site. More specifically, exposure setting characteristics include climate and weather conditions, vegetation, ground-water hydrology, surface water identification, soil characteristics and population characteristics such as location relative to the site, human activity patterns, and the location of any sensitive subpopulations.

Step 2: Identification of exposure pathways. Exposure pathways are based on the types and location of chemicals present at the site, the sources and release mechanisms of these chemicals, the likely environmental transport and fate of these chemicals, and the location and activities of populations that may be exposed. For each exposure pathway, exposure points and routes of exposure are identified.

Step 3: Quantification of exposure concentrations. This step involves three stages. During the first stage, the exposure assessor determines the concentration of chemicals that will be contacted, the frequency of the contacts, and the duration of each contact. Monitoring data combined with chemical fate and transport models are used to estimate the exposure concentrations. Both current and future exposure concentrations are estimated. The assessor also calculates chemical intakes,



or “exposure estimates.” This done for each exposure pathway identified in Step 2. Chemical intakes are expressed in terms of the mass of the contaminant in contact with the body per unit body weight per unit time. After intakes have been estimated, they are organized by grouping all applicable exposure pathways for each exposed population. Sources of uncertainty in the exposure estimates must be evaluated because this may affect decisions regarding the degree of remediation necessary at the site. Actions at Superfund sites should be based on the level of exposure reasonably expected to occur under both current and future land use conditions.

#### 1.2.4 Toxicity Assessment

Toxicity assessment is the third component of a baseline risk assessment. During a toxicity assessment, risk assessors establish whether a substance can potentially produce an adverse effect (“hazard identification”), and quantify the dose/response relationship (“dose/response evaluation”). Hazard identification establishes whether exposure to an agent can cause an increase in the incidence of a particular adverse health-effect (e.g., cancer, birth defects) and whether the adverse health effect is likely to occur in humans. During the dose/response evaluation, assessors evaluate the toxicity information and quantitatively characterize the relationship between the dose of the contaminant or amount of exposure received by people or animals and the incidence of adverse health effects in the exposed population. Toxicity values are derived from the dose/response evaluation and are used in the next step (risk characterization) to estimate risks at different exposure levels.

The first part of the hazard identification process involves the collection of toxicological information. Assessors collect data from a variety of sources, including epidemiologic studies, clinical studies, and animal experiments. Information may also be available from supporting studies such as *in vitro* experiments. The most convincing evidence of human risk derives from epidemiological studies showing an association between an agent and a disease. Human toxicity studies are given first priority in the dose-response assessment if they are available, and animal studies are used as supportive evidence. Adequate human studies are usually not available; therefore, animal studies are most often used.

Metabolic and pharmacokinetic studies, cell cultures, and structure-activity relationship studies are used to support conclusions about the likelihood of occurrence of adverse health effects in humans. Metabolic and pharmacokinetic studies may provide insights into the mechanism of action of a particular compound exhibiting a toxic effect. Studies using cell cultures or microorganisms may be used to estimate a compound’s potential for biological activity. Structure-activity studies may predict toxicologic activity based on analysis of chemical structure.

EPA recommends that risk assessors use the Integrated Risk Information System (IRIS) as the primary source for toxicity data. IRIS contains only those reference doses (RfDs) and cancer slope factors that have been verified by EPA experts. The Health Effects Assessment Summary Table (HEAST) (USEPA, 1994) presents toxicity information and values for which Health Effects Assessments (HEAs), Health and Environmental Effects Profiles (HEEPs), Health Assessment Documents (HADs), or Ambient Air Quality Criteria Documents (AAQCDs) have been prepared. HEAST summarizes interim and verified RfDs and slope factors, and refers risk assessors to the most current sources of supporting toxicity information. HEAST is a good source to supplement IRIS.



EPA developed the reference dose to express, within an order of magnitude, daily exposure levels for specific noncarcinogenic chemical substances (carcinogenic substances are handled differently, as described below). Three types of RfDs have been developed: chronic, subchronic, and developmental. Chronic RfDs are developed for exposures between seven years and a lifetime, subchronic RfDs for exposures between two weeks and seven years, and developmental RfDs for single exposures during gestation. EPA believes that for chronic RfDs daily exposures below the RfD are unlikely to cause “appreciable risk of deleterious effects during a Lifetime” (USEPA, 1989).

The experimental study used to calculate the RfD is called the critical study. It is chosen by ascertaining either the test species that is the most relevant to humans based on a biological rationale or the species that demonstrates a toxic effect at the lowest dose. After dosimetric conversions are made to the animal data to adjust for species differences, the critical toxic effect is chosen. This is done by determining the dose that results in the lowest-observed- adverse-effect-level (LOAEL) for any noncancer adverse effect. Next, the highest experimental dose level that does not produce an adverse effect is determined from the critical study. This value is called the no-observed-adverse-effect-level (NOAEL), and it is the key data point used to calculate the RfD. If the NOAEL is not available, then the LOAEL can be used to calculate the RfD. *{Note: RfDs are calculated for oral exposures. Reference concentrations (RfCs) are calculated for inhalation exposures. They are both derived in the same manner.}*

The RfD is calculated by dividing the NOAEL (or LOAEL) for the critical toxic effect by uncertainty factors and a modifying factor. Each uncertainty factor represents a specific area of uncertainty inherent in extrapolating data from experiments to expected real-life scenarios. The modifying factor reflects a qualitative, professional assessment of additional uncertainties in the entire data base for the chemical not explicitly addressed by the uncertainty factors. In general, the modifying factor (MF) reflects the overall confidence that the EPA experts have in the experimental data.

As already mentioned, carcinogens are handled differently. Scientists believe that a small number of certain molecular events can cause cellular changes that may lead to carcinogenesis. According to EPA, exposure to any amount of chemical or agent that is capable of producing these effects has a finite probability, however small, to initiate this process. Therefore, there is no exposure level that is considered to be totally risk free, and a threshold cannot be estimated for carcinogens.

EPA uses a two-part process to evaluate carcinogens. The first part is to assign a “weight of evidence” to the data to determine the likelihood that the substance is actually a human carcinogen. The weight of evidence places the chemical or agent into one of five groups as shown in Table 1.1 (USEPA, 1989). The second part is to calculate a chemical-specific slope factor. Cancer slope factors determine the potential risk levels associated with exposure to carcinogens. First, the assessor chooses an appropriate dose/response data set from either animal or human data. Then, a high-to-low dose extrapolation model is applied to the data set(s). The model is used to extrapolate from the high doses utilized in experiments to the low doses expected to be found in the environment (i.e., exposure levels). The slope factor is calculated by taking the upper 95th percent confidence limit of the slope of the line from the model fitted to the dose/response curve. This value represents a 95-percent probability of a response per unit intake of a chemical over a lifetime. In other words, there is only a 5-percent chance that the probability of the response will be higher.



Table 1.1: EPA Weight-of-Evidence Classification System for Carcinogenicity

Group	Description
A	Human carcinogen
B1 or B2	Probable human carcinogen <i>B1 indicates that limited human data are available</i> <i>B2 indicates sufficient evidence in animals and inadequate or no evidence in humans</i>
C	Possible human carcinogen
D	Not classifiable as to human carcinogenicity
E	Evidence of noncarcinogenicity for humans

### 1.2.5 Risk Characterization

Risk characterization, the final component of the baseline risk assessment, combines the toxicity and exposure assessments into quantitative and qualitative expressions of risk. There are six major steps in risk characterization (USEPA, 1989), as described below:

Step 1: Organize outputs of exposure and toxicity assessments

Step 2: Quantify pathway risks

Step 3: Combine risks across pathways that affect the same individuals over the same time periods

Step 4: Assess and present uncertainty

Step 5: Consider site-specific health or exposure studies

Step 6: Summarize results of the baseline risk assessment

Activities carried out in each of these steps are briefly described in the following sections,

#### 1. Organize Outputs of Exposure and Toxicity Assessments



Before the final risk calculations are begun the assessor should verify that all necessary exposure and toxicity information has been collected and organized for each exposure pathway and land use evaluated. The exposure periods (e.g., short-term, chronic) for the toxicity and exposure values must be the same. For example, cancer risks should always be expressed as average lifetime exposure. Less-than-lifetime exposure values should be converted to average lifetime exposure. Chronic RfDs should only be compared with long-term (i.e., greater than 7 years) exposures. Subchronic noncarcinogenic exposures can not be converted to chronic exposures. Subchronic exposures should only be compared with subchronic RfDs.

Exposure routes should be properly matched for the toxicity and exposure values (e.g., oral to oral, inhalation to inhalation). Toxicity values for localized effects (e.g., dermal) should not be compared to systemic exposures (e.g., oral, inhalation). Conversion of values from one exposure route to another (e.g., oral to inhalation) should only be done after consulting with EPA's Environmental Criteria and Assessment Office (ECAO). The units used to express toxicity and exposure values for the inhalation exposure route should be checked to make sure that they are identical. Inhalation values can be expressed either as mg/m<sup>3</sup> or as mg/kg-day.

Finally, both the toxicity value and exposure estimate should be expressed either as absorbed dose or intake (i. e., administered dose). Except for dermal exposures, the preferred method is to express these values as intakes. Dermal values must take into account absorption through the skin. If strong evidence is available for absorption efficiencies for the oral and inhalation routes, then the toxicity and exposure values may be appropriately modified.

## 2. Quantify Pathway Risks

### A. Calculate Risks for Individual Substances

The first step in assessing composite risks is to calculate risk for individual substances. "For carcinogens, risks are estimated as the incremental probability of an individual developing cancer over a lifetime as a result of exposure to the potential carcinogen" (USEPA, 1989). Because chemical intakes at Superfund sites are relatively low compared to doses given to experimental animals, it is assumed that these intakes lie in the linear portion of the low-dose, multistage dose-response model. This assumption results in the slope factor being a constant, and therefore the risk is directly related to the exposure intake. The carcinogenic risk equation is (USEPA, 1989):

$$\text{Risk} = \text{CDI} \times \text{SF}$$

Where:

Risk = a unitless probability (e.g.,  $2 \times 10^{-5}$ ) of an individual developing cancer

CDI = chronic daily intake averaged over 70 years (mg/kg-&y)

SF = slope factor, expressed in (mg/kg-day)<sup>-1</sup>

Currently, EPA does not use probabilistic approaches to estimate the potential for noncarcinogenic health effects. Instead, a noncancer hazard quotient (HQ) is calculated for noncancer health effects:





$$\text{Noncancer Hazard Quotient} = E/RfD$$

Where:

E = exposure level (or chemical intake)

RfD = reference dose for same exposure pathway as E

This ratio does not represent a statistical probability of an adverse effect occurring. The hazard quotient assumes that for noncancer health effects a threshold dose exists below which adverse health effects are unlikely to occur, even for sensitive populations. If the hazard quotient is less than unity there is less concern for adverse health effects. The higher the hazard quotient is above unity, the greater the concern.

### B. Aggregate Risks for Multiple Substances

The second step in assessing risks for each pathway is to calculate risk for multiple chemicals or chemical mixtures. When little or no quantitative information is available on the potential interaction among the components in a chemical mixture, EPA recommends dose additivity (USEPA, 1989; USEPA, 1992). For carcinogens, assessors assume a linear dose-response curve; therefore, they calculate total carcinogenic risk by adding the estimated carcinogenic risk for each substance of concern. For carcinogens, addition of individual risks assumes three things: that the various carcinogens in the mixture act independently, that intakes of the individual substances are small, and that all of the substances produce the same effect. Summed cancer risk estimates should be expressed using only one significant figure.

Risks from mixtures of noncarcinogens are assessed differently. For these, assessors calculate a hazard index (HI) for each exposure pathway. This is done by adding the individual HQs for that pathway. HQs should only be summed for those chemicals that produce the same toxic effect (that is, those chemicals that affect the same target organ) and that do so by the same ‘mechanism of action and for the same exposure period (chronic, subchronic, and shorter-term exposures). “This approach assumes that simultaneous subthreshold exposures to several chemicals could result in an adverse health effect” (USEPA, 1989). It also assumes that the magnitude of the adverse effect will be proportional to the sum of the hazard quotient ratios and that 100 percent of the contaminant is absorbed for each exposure pathway.

### 3. Combine Risks Across Exposure Pathways

The final step in the risk estimate is to sum the risks across exposure pathways, if appropriate. Some individuals might be exposed to a substance or combination of substances through several pathways. By following the steps below, assessors ensure that the estimate for the total site risk is appropriate.

- First, assessors identify the reasonable exposure pathway combinations. For each pathway, cancer risks and HIs are developed for particular exposure areas and time periods. If two pathways do not affect the same individual or subpopulation, neither pathway’s individual cancer risk estimate or HI affects the other, and they should not be combined.



- Second, assessors examine the likelihood that the same individuals would face the reasonable maximum exposure (RME) by more than one pathway. Because contaminant concentrations vary over time and space, one individual may not experience the RME for more than one pathway over a certain period of time. Therefore, assessors may combine the RME risks for more than one pathway if they can explain why the key RME assumptions for these pathways apply to the same individual or subpopulation.

#### 4. Assess and Present Uncertainty

Highly quantitative, statistical uncertainty analysis is usually not necessary for Superfund risk assessments. It is more important to identify the site-related variables and assumptions that contribute to the uncertainty rather than to precisely quantify the degree of uncertainty in the risk assessment. The categories of uncertainties associated with risk assessments include the following:

- initial selection of substances,
- toxicity values for each substance, and
- exposure assessment for individual substances and individual exposures.

#### 5. Consider Site-Specific Health or Exposure Studies

The results of the BRA should be compared with the results of the Agency for Toxic Substances Disease Registry (ATSDR) health assessment. This is a separate assessment conducted by the ATSDR. It is more qualitative in nature, and focuses on sensitive populations, toxic mechanisms, and possible public health impacts associated with exposures at a site. It is a source of additional information for the risk assessor. If there are any differences between these studies the reasons for these differences should be explained in the BRA. The assessor should also check with local health agencies and ATSDR to determine if any site-specific epidemiological studies have been performed. Any existing studies should be reviewed carefully by a trained epidemiologist to check for confounding factors not associated with the site. The results of the BRA and any reliable site-specific study should be compared and explained in the risk characterization section of the BRA.

#### 6. Summarize and Present the Baseline Risk Assessment Results

Presentation of the risk assessment should address the following (USEPA, 1989):

- a discussion of the degree of confidence that the key site-related contaminants were identified, and their concentrations relative to background determined;
- a description of the various types of health endpoints (i.e., cancer, noncancer) present at the site, and a discussion distinguishing between those effects known to occur in humans and those that are predicted to occur based on animal experiments,



- a discussion of the level of confidence in the quantitative toxicity information, and a discussion of qualitative toxicity information on the substances not included in the quantitative assessment;
- a discussion of the level of confidence in the exposure assessment
- a comparison of the site cancer risk and noncancer hazard index values to the Superfund site remediation goals in the NCP (e.g., the cancer risk range of  $10^{-4}$  to  $10^{-7}$  and noncancer hazard index of 1.0);
- a discussion identifying the major factors driving the site risks (e.g., substances, pathways, and pathway combinations);
- a discussion of the major factors contributing to the uncertainty in the risk assessment (e.g., adding risks over several substances and pathways);
- a characterization of the exposed population; and
- a comparison of the site BRA results with ATSDR site-specific health studies, when available.

A tabular summary of the estimated cancer and noncancer risks should be prepared for all exposure pathways and land uses analyzed and for all substances carried through the risk assessment.

### **1.3 How to Use This Document**

Detailed discussions of baseline risk assessment issues related to eight key policy topics are provided in Chapters 3 through 10. The topics addressed in those chapters are as follows

- Exposure and Dose - Chapter 3,
- Estimation of Average and Upper-End Risk - Chapter 4,
- Chemical Mixtures - Chapter 5,
- Radiation Risk Assessment - Chapter 6,
- Noncancer Health Endpoints - Chapter 7,
- Institutional Controls in Baseline Risk Assessments - Chapter 8,
- Site Specific Data vs. Default Values - Chapter 9, and



- Data Gaps, Uncertainty, and Professional Judgement - Chapter 10.

Each chapter begins with an introduction that explains how the topic/issues fit into the overall analytical framework of the baseline risk assessment process. Next, an historical perspective on how the topic has evolved is provided. The statutory and regulatory origins of the topic are detailed, and relevant guidance that has been developed addressing the topic are identified and reviewed. It is within these discussions that specific issues pertaining to the topic are identified and analyzed. The evolution of each issue will be presented chronologically. The major development, changes, etc. in the issue, its underlying science policy, and/or its application to the baseline risk assessment process will be highlighted. The current status of and position on each issue will be detailed, along with the issue's pros, cons, and uncertainties. The potential impacts on the outcome of risk assessments and the basis for negotiation will be clearly presented. In addition, each chapter will outline conclusions as to how those issues affect the interpretation of estimated baseline risks, and how they may provide a basis for negotiation with EPA. Finally, each chapter concludes with a listing of the reference materials cited in that chapter's text.

Discussion of the statutory history of each topic, its evolution, etc., is presented fully within each chapter. Thus, readers interested in only a specific topic can turn to the appropriate chapter and find a full discussion of pertinent information relating to that topic without having to cross-refer to other chapters. Readers of this entire document will observe, however, that in order to treat the subject matter in each chapter completely, a certain amount of unavoidable repetition of information that pertains to multiple topics exists from chapter to chapter.

Chapter 2 is structured differently from the other chapters in that it directs the reader to detailed information relating to specific risk assessment issues to be found in Chapters 3 through 10. The introduction to Chapter 2 illustrates the correspondence between each chapter and the specific component of the risk assessment process to which it pertains. Subsequent sections of Chapter 2 provide an overview of the specific issues to be evaluated under the risk assessment topics addressed in Chapters 3 through 10. Finally, the chapter concludes with an index of key risk assessment terms.

Appendix A, which follows Chapter 10, contains a brief summary of each of the source documents used in developing this guidance. It is intended to constitute a resource for identifying sources of information necessary for those who are responsible for developing or evaluating baseline risk assessments.

## **1.4 References**

CERCLA. 1980. Comprehensive Environmental Response, Compensation, and Liability Act. PL 96-510.

SARA. 1986. Superfund Amendments and Reauthorization Act. PL 99-499.

USEPA. 1985. National Oil and Hazardous Substances Pollution Contingency Plan. U.S. Environmental Protection Agency. [50 FR 47912].

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USEPA. 1992. Guidelines for Exposure Assessment. U.S. Environmental Protection Agency, Office of Health and Environmental Assessment. Washington, D.C. [57 FR 22888].

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